Severe Skin Necrosis in Adult: an Unusual Complication of Fulminant Meningococcal Sepsis

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SUMMARY We report on a 20-year-old female high-school student with fulminant meningococcal sepsis presenting as severe skin necrosis in lower extremities. The wound surface affected 2% of her total body surface area and was covered with black eschar. The adipose tissue beneath the eschar was degenerated and turned to necrosis as to deep fascia and periosteum of lower tibia. Excision of necrotic eschar, then covered with flaps on both legs resulted in a successful outcome.

KEY WORDS: skin necrosis, fulminant meningococcal sepsis, adult, excision, flap

INTRODUCTION

Meningococcal meningitis is a severe life-threatening disease caused by the bacterium Neisseria (N.) meningitidis, a gram-negative, aerobic, and encapsulated diplococcus. It most commonly affects children under 5 years of age. The secondary incidence peak is among adolescents, particularly those who live in crowded dormitories (high-school students, college freshmen and military recruits) (1). Meningococcal infection occurs year-round, with predominance in winter and early spring. Despite effective antimicrobial and supportive therapy, meningococcal disease remains a major infectious cause of death. The overall case-mortality rate approaches 10%-14%, even if prompt medical treatment is administered (2). Devastating presentations such as fulminant meningococcal sepsis can even lead to death in an otherwise healthy young person in less than 48 hours. Beyond high mortality, up to 20% of survivors suffered lifelong sequels, such as hearing loss, brain damage, loss of limb, severe scarring, and psychological problems including anxiety, depression, etc. (3).

Severe skin and soft tissue loss is rare in meningococcal septicemia of adults. This is usually due to systemic vasculitis, characterized by endothelial damage with increasing capillary permeability and eventually occlusion of microvascular circulation resulting from direct infection by N. meningitidis and immune reaction of endotoxin. At autopsy, a fibrin-leukocyte-platelet clot forms within the vessels, particularly the venules of necrotic purpuric skin, and obstructs blood flow (4,5).

The case we present herein illustrates such a phenomenon in a female high-school student who
developed severe skin necrosis in lower extremities secondary to fulminant meningococcal sepsis. The wound repair and reconstructive process are described.

CASE REPORT

A previously healthy 20-year-old female presented to a local emergency room with a 1-day history of nausea, emesis, non-bloody diarrhea and hyperpyrexia with temperature of 39.2 °C and then was admitted to the Infection Department with symptoms of somnolence, fever, hypotension and tachycardia. Physical examination was significant for profound acrocyanosis and a diffuse purpuric rash blister on her arms, legs and trunk. Laboratory investigations included hemoglobin 10.7 mmol/L⁻¹, white cell count 11.08×10⁹/L⁻¹, platelet 24×10⁹/L⁻¹ and prothrombin time 37.1 second. Then a dramatic progression from petechiae to confluent ecchymoses was observed on her skin, apparently fitting the clinical profile of disseminated intravascular coagulation (DIC), and fulminant meningitis was suspected. Prompt bacteriological diagnosis was performed. Lumbar puncture and skin biopsy were performed upon admission. Gram stain of the cerebrospinal fluid (CSF) and initial blood cultures identified gram-negative diplococci and \( N. meningitidis \) was also isolated. In addition, skin lesion specimens were positive for gram-negative diplococci. Consequently, the patient was diagnosed with fulminant meningococcal meningitis, septicemia and septic shock, placed in isolation and intravenous antibiotic (ceftriaxone 2g Q12 h) was started immediately and continued for 8 days.

After treatment with steroids and anticoagulants, most of purpuric ecchymoses on the trunk and both lower extremities healed spontaneously. However, the ecchymoses located on both medial malleoli persisted, showing a confluent pattern, with purulent secretion emerging around them. These were reviewed by the burns surgery team and were initially treated conservatively with silver sulfadiazine coverage only. Debridement and skin grafting were considered to be necessary eventually, but were delayed at this stage for hemodynamic instability.

On day 40, the patient recovered from this severe episode and was transferred to Burns Unit with severe cutaneous necrosis as a manifestation (Fig. 1). In addition, many coin-sized punctiform skin lesions developed on both anterior tibiae. The wound surface affected was estimated at 2% of total body surface area (TBSA) and was covered with black eschar. Examination of the wound revealed that adipose tissue beneath the eschar was degenerated and turned to necrosis as to deep fascia and periosteum of lower tibia. \( Staphylococcus aureus \) was detected in deep wound tissue. On day 47, she underwent debridement of necrotic eschar on both legs, consequently with bilateral medial malleolus joints exposed. Additionally, the damaged left tendo calcaneus and gastrocnemius fascicle were also excised.

After the procedure of dressing changes twice a day, wound infection was assessed well-controlled and the areas of skin loss in the right lower extremity were covered by transfer of the posterior tibial artery perforator (PTAP) flap. Forty-two days after debridement, digital subtraction angiography (DSA) examination revealed that collateral circulation of the left posterior tibial artery decreased obviously above the medial malleolus (Fig. 2). A comparatively large perforator branch at 13 cm above existed. Then the pedicle of the posterior tibial artery perforator flap was designed by DSA presentation and segregated to cover the left medial malleolus wound. Unfortunately, the remnant left tendo calcaneus was completely bro-
ken down for inappropriate weight-loading and left medial malleolus was exposed again. A cross-leg flap from the right lower extremity was used to cover the wound. Three weeks later, the pedicle division was performed until the patient was discharged. She was followed up accordingly and progressed well with rehabilitation support. Nine months after admission, she could walk with orthopedic footwear (Fig. 3).

DISCUSSION

*Neisseria meningitidis* is a gram-negative organism, often encapsulated diplococcal in form, which was first isolated in 1887. Six serogroups (serogroups A, B, C, W-135, X and Y) are the major pathogens involved in causing acute, life-threatening meningococcal infections worldwide (6). About 5% to 25% of patients with meningococcal infection develop purpura fulminans (PF) (7). The incidence of PF from *N. meningitidis* is certainly higher in children and adolescents, while being very rare in adults. Bollero *et al.* reviewed only 19 adult cases of PF in meningococcal septicemia in the literature during the 1997-2009 period, and report the experience in the management of skin infarction after meningococcal septicemia in the sole adult patient among five patients (8). PF associated with extensive skin and soft tissue necrosis is a rare and severe complication of meningococcal septicemia. To our knowledge, this is the first reported case of severe skin necrosis in an adult, which resulted from fulminant meningococcal sepsis.

Hemorrhagic skin lesion is characteristic of fulminant meningococcal septicemia with initially petechial rash, rapidly progressing to confluent purpura. Skin changes may occur in any part of the body and the areas such as the anterior tibia and medial malleolus are at greatest risk due to not much subcutaneous fat or muscle. If progressing rapidly, meningococcemia can lead to extensive cutaneous infarction often associated with necrosis of underlying fat and muscle or even with severe subsequent gangrene. The basic
physiopathologic effect on the skin is DIC, characterized by meningococcal endotoxin-induced edema formation and capillary thrombosis. Several precautions such as appropriate wound care and management have been recommended to prevent unwanted damage to tissue at risk (9).

The similarity between skin necrosis secondary to meningococcal septicemia and full-thickness skin burns provides an optimal treatment in burn unit. In this patient, the wounds were treated with topical application of silver sulfadiazine during the initial, critical phase to prevent bacterial colonization and infection. When the patient recovered from septicemia and the vital signs remained stable, she was transferred to the burn unit for definitive treatment.

Skin grafting is the mainstay of burn surgical management for the majority of cases where there have been skin necrotic lesions resulting from purpura fulminans. However, in this case, the injury wound located in both medial malleolus was excised and the tendons and bone were exposed after first debridement. There are many treatment options for reconstruction of leg and foot deep defects with exposure structure such as bone, joint and tendon. One alternative includes local flaps. PTAP flap is acknowledged as the best donor site resurfacing the medial malleolus without microvascular anastomoses and with minimal donor-site morbidity (10,11). Anatomical studies show that posterior tibial artery perforators are consistently found to be the largest in the lower leg within three 5-cm intervals: 4 to 9 cm, 13 to 18 cm, and 21 to 26 cm from the intermalleolar line (12). It is difficult to know clinically how far a zone of microvascular damage adjacent to necrotic lesions extends in N. meningitidis-induced septic emboli. Hence, Doppler and DSA monitoring of microvascular areas is necessary to recognize vascular compromise early enough to result in an overall flap success.

In this case, there was a 4×4 cm² defect area in the right medial ankle region. A perforator of the posterior tibial artery at 5 cm above the tip of the right medial malleolus was detected by hand-held Doppler. The wound was thoroughly debrided and repaired with fasciocutaneous PTAP flap based on distal perforators. Preoperative DSA study showed the lowest perforators of the posterior tibial artery at 13 cm from the left intermalleolar line. The extensive left defect was subsequently treated with distant PTAP flap based on middle zone perforators. Nonetheless, the purulent secretion induced by multiple-resistant \textit{Staphylococcus aureus} (MRSA) appeared underneath the right PATP flap and flap necrosis was ultimately presented. In addition, an accidental weight-load-


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